BRITISH MEDICAL BULLETIN

Vol. 1. (1943) No. 9

The object of this Bulletin is to provide a guide to medical science and thought in Britain, and it consists mainly of summaries of a representative selection of British papers on subjects of medical interest. Any material appearing in the Bulletin may be published without fee, but acknowledgment of the source, by addition of the initial letters BMB followed by the serial numbers of the items selected, would be appreciated.

SPECIAL CONTRIBUTIONS

THE DEVELOPMENT OF OPHTHALMOLOGY IN BRITAIN
by Mr. R. R. James ... 100

THE TASK OF OPHTHALMOLOGICAL RESEARCH
by Professor A. Sorsby ... 102

OPHTHALMOLOGY

Statistical Analysis of Persons Certified Blind ... 104
Anomalous Forms of Amaurotic Idiocy ... 104
Ætiology of Phlyctenular Ophthalmia ... 105
Experimental Hypopyon Ulcers ... 105
Sulphonamides in Experimental Ocular Infections ... 105
Avascular Healing in the Cornea ... 106
Removal of Magnetic Foreign Bodies ... 106
Vitamin A and Dark Adaptation ... 106

STOMACH IN HEALTH AND DISEASE

Gastric Motility in Man ... 107
Gastric Acidity in the First Year ... 107
Evaluation of Gastroscopy ... 107
Polyps and Polypoid Gastritis ... 108
Hæmatemesis and Melæna ... 108

CARDIOVASCULAR SYSTEM

Signs Simulating those of Mitral Stenosis ... 109
Cardiac Symptoms Complicating Diabetes ... 109
Blood Pressure and Old Age ... 110

LIBRARY
MEDICAL FACULTY
McGILL

SPECIAL CONTRIBUTION

[This article is the first of a series of brief outlines of the development of special aspects of Medicine in Britain. The articles will be published occasionally as they are received, and the order of publication does not therefore follow a rational plan.
The object of the series is to provide, for those whose interest in medical work in Britain is of relatively recent origin, a background to the contemporary observations which are reported in British Medical Bulletin.]

THE DEVELOPMENT OF MEDICAL STUDIES IN BRITAIN: I. OPHTHALMOLOGY

by R. R. JAMES, F.R.C.S.

Consulting Ophthalmic Surgeon to St. George's Hospital, London
Senior Editor of the British Journal of Ophthalmology

Earliest Times

English ophthalmological history may be said to start with the Roman occupation of Britain. From the large number of oculists' stamps which have been unearthed on Roman sites we know that a good deal of local treatment of eye conditions by way of collyria and ointments was practised at this early date. After the withdrawal of the legions there is a long gap in our knowledge; in fact nothing is known until we come to the Anglo-Saxon *Leech* [medical] books and Herbals. Here again, treatment was mainly by local applications of infusions of herbs and the secretions of animals, such as gall mixed with honey, and even of human urine. Charms also played a large part and we may say that Anglo-Saxon ophthalmology has every appearance of having been largely futile.

The Norman Conquest and After

The Norman Conquest did little to improve the practice of ophthalmology but the 13th century saw the beginnings of optics in Britain. Grosseteste, Bishop of Lincoln, was the teacher of Roger Bacon (died 1294), who proved that spherical *plus* spheres would be of use for reading in old people; while John of Peckham (died 1294), later Archbishop of Canterbury, is credited with the discovery of the use of concave refracting surfaces, and his *Perspectiva Communis* was for the next few centuries the only text-book on optics to be used in England.

In the year 1377 John of Arderne wrote his little book entitled *De cura oculorum*, of which manuscripts in Latin and in 15th-century English exist among the *Sloane Manuscripts* at the British Museum, and in the Library of Emmanuel College, Cambridge. This booklet is a mere compilation of other people's views, much of it being taken from Lanfranc. English ophthalmology had, in fact, made very little progress since Anglo-Saxon times.

The Sixteenth and Seventeenth Centuries

The closing years of the 16th century saw the publication of two small books dealing with ophthalmology. One was Walter Bayley's *Briefe Treatise Touching the Preservation of the Eye-sighte*, and the other a translation (probably by A. Hunton, of Newark-on-Trent) of Guillemeau's *Maladies de l'Œil*.

Early in the 17th century Richard Banister, of Stamford, brought out a duodecimo of 478 pages containing five separate treatises. Of these the first 112 pages are Banister's own contribution, and this section is named Banister's Breviary. Though he was an itinerant oculist it is obvious that Banister was a much more honest practitioner than the rest of his ophthalmic contemporaries. He it was who first pointed out the cardinal sign of hardness of the eyeball in cases of *gutta serena*, in this case glaucoma. The significance of his observation was not recognised, and raised intraocular tension was forgotten for the next 150 years. Banister was also almost certainly the author of the manuscript in the British Museum (*Sloane Manuscript*, 3801) which throws much light on the manners and customs of the itinerant charlatans of this date. Several of these are named, such as Luke of Erith, Mr. Surphlete of King's Lynn, and Henry Blackborne. The last-named is severely criticised by Banister, and it is a sign of the looseness of the times that Blackborne in 1605 received the Archbishop of Canterbury's licence to practise in diseases of the eyes. Richard Banister must have been a remarkable man and has deserved well of ophthalmology.

Turberville of Salisbury (died 1696) was a qualified medical man in an age of unqualified quacks. He had a large practice and made a valuable contribution to ophthalmology in extracting an iron particle from the cornea with a magnet. Another qualified English physician of this time was William Briggs, who published the *Ophthalmographia* and the *Nova Visionis Theoria*. He described the papilla of the optic nerve in 1676 and, in 1684, recorded in the *Philosophical Transactions of the Royal Society* a case of night-blindness. Dimness of vision following the administration of Peruvian bark in fever was described in 1681 by a general practitioner at King's Lynn in a letter to Briggs.

The Eighteenth Century: Charlatinism and the Foundations of Modern Ophthalmology

The 18th century was the age of ophthalmic quackery, but it saw the foundations of modern ophthalmology laid by such masters as Cheselden, Sharp and Warner. Cheselden devised the operation of iridotomy in the making of an artificial pupil, and Sharp and Warner perfected the operation of extraction of cataract, a great advance on the age-long method of couching [displacement of the cataract from the visual axis].

Duddell, a pupil of Woolhouse, wrote a good account of diseases of the cornea in 1729. Later in the century, James Ware wrote on syphilis in connexion with ophthalmia, and brought some order out of chaos in the matter of conjunctival diseases. Gataker wrote on the anatomy of the eye and on the use of belladonna, and Wells on double vision.

The chief 18th-century ophthalmic quacks were William Read, Roger Grant, and the Chevalier Taylor. Read and Grant were illiterate; the former was knighted by Queen Anne. The Chevalier Taylor was quite a remarkable person. In professional knowledge he was often far ahead of his time, but he practised all the arts of unblushing effrontery and charlatanism. He was oculist to George II, and his son and grandson followed in his footsteps though they were not of the same calibre. Eighteenth-century Royalty was singularly unfortunate in its oculists, as George Coats pointed out.

The end of the century, in 1794, saw the publication by John Dalton of the history of his colour blindness. Thus, it will be seen that up to the end of the 18th century ophthalmology had not advanced very far, but better times were to come.

Rational Ophthalmology Displaces Charlatanism

The year 1805 saw the foundation of **Moorfields* Eye Hospital by J. C. Saunders, and this more than anything else struck the death blow to the quackery of the previous century. Provincial Eye Hospitals were founded at Exeter in 1808, Bristol in 1810, and Manchester in 1814. The *Royal Westminster* Ophthalmic Hospital was founded by Guthrie in 1816, the *Central London* Ophthalmic Hospital in 1843, the *Western* Ophthalmic Hospital in 1856 and the *Royal* Eye Hospital in the year following. The first course of lectures on diseases of the eye was given by Guthrie at the *Royal Westminster* Ophthalmic Hospital.

An earlier epoch-making date in ophthalmological history is 1801, when Thomas Young published his paper on the mechanism of the human eye in the *Philosophical Transactions*. He described astigmatism and measured the amount of the astigmatism in his own eye. His table of optical constants has been only very slightly modified by modern research. His theory of colour vision postulated the presence in the

* [Now the *Royal London* Ophthalmic Hospital.]

retina of three "fibres," which correspond to the colours red, green and violet respectively. This theory was later resuscitated by Helmholtz and is known as the Young-Helmholtz theory. Young's experiments on interference strongly supported the undulatory theory of light already adumbrated by Sir Isaac Newton and Huygens.

Ophthalmological Literature and Societies

The first real text-books of ophthalmology in Britain belong to the first half of this century. In 1830 William Mackenzie of Glasgow brought out his great work on diseases of the eye. It was far ahead of any previous text-book on the subject and it ran to a fourth edition. Mackenzie was a master clinician, and was the first surgeon to give an adequate account of sympathetic ophthalmitis. Sir William Lawrence's text-book appeared in 1833, and Richard Middlemore's in 1835. Before this, Travers had brought out his synopsis of diseases of the eye in 1820. It ran to a third edition. J. C. Saunders published a book before his untimely death, and did much to revolutionize the treatment of congenital cataract by insisting on early discission. In this he followed the practice of Woolhouse, an English surgeon, who was resident in Paris for many years in the previous century.

Wardrop's *Essay on the Morbid Anatomy of the Human Eye* laid the foundations of ophthalmic pathology; Tyrrell's *Diseases of the Eye*, in two volumes, appeared in 1840; and Dalrymple's splendid atlas belongs to about this date.

The year 1881 saw the foundation of the *Ophthalmological Society of the United Kingdom* with Sir William Bowman as first president. It grew out of informal discussions in the house-surgeon's room at old *Moorfields*, and of the Committee appointed to make arrangements for its foundation Sir Thomas Barlow is the sole survivor. Sir H. Lindo Ferguson of New Zealand is also still with us as an original member of the Society. The Society now has, as affiliated members, the *Oxford Congress*, founded by R. W. Doyne, the *North of England Ophthalmological Society*, founded by Percival Hay and J. Gray Clegg, the *Midland and South Western* Societies, the *Irish Ophthalmological Society*, and the *Scottish Ophthalmic Club*. A volume of *Transactions* has been published each year without a break since 1881, and the series forms a rich mine of ophthalmological facts.

In 1857 the staff at *Moorfields* began the publication of the invaluable *Ophthalmic Hospital Reports*, which ran to 20 volumes. In 1864 J. Z. Laurence and T. Windsor began the publication of the old *Ophthalmic Review*. It came to an untimely end 3½ years later and the new *Review* was started in 1881 by Priestley Smith of Birmingham and Karl Grossmann of Manchester. The *Ophthalmoscope* was founded by Sydney Stephenson in 1903. In 1917 the first number of the *British Journal of Ophthalmology* appeared. It incorporates the *Moorfields' Reports*, the *Ophthalmic Review* and the *Ophthalmoscope*, and has continued without a break to the present day.

Ophthalmic Instruments and Operations

English ophthalmology has been responsible for many inventions of ophthalmic instruments. It should not be forgotten that William Porterfield (died 1771) devised the first optometer. Charles Babbage, in 1848 constructed an opthalmoscope which left little to be desired. He showed it to Wharton Jones, who, alas! did not realize the importance of the means of research thus placed at his disposal. Babbage was a mathematician, not an ophthalmic surgeon, and finding that Wharton Jones was not interested in his model he took no further steps and it was left to Helmholtz to bring out his instrument in 1851. Tyrrell devised the iris hook which bears his name; Bowman a trephine for the eye-ball; Mules of Manchester first suggested the insertion of a glass globe in the sclerotic after evisceration of the eyeball, and his practice was later modified by Frost and Lang, who inserted globes of glass or metal into the orbit.

Corneo-sclerotic trephining for chronic glaucoma was first proposed by Freeland Fergus of Glasgow in 1909. In the next year Elliot, of Madras, improved the operation by splitting the superficial layers of the cornea in order to make more sure of tapping the anterior chamber, and the operation has been known by his name ever since. Herbert, of the Indian Medical Service, also made important additions to our anti-glaucoma methods at this date.

Stanford Morton's ophthalmoscope was for years the best of its kind, though Frost's instrument was a close second. Frost also brought out an extremely effective model eye for teaching purposes.

Ophthalmology as a Speciality

In the early years of the last century ophthalmology was still, in Britain, a part of General Surgery; Sir William Bowman was the first general surgeon to give up his surgical work at *King's College* Hospital in order to become a pure ophthalmic specialist. With George Critchett, Bowman was responsible for the foundation of this speciality. But Critchett, and even Jonathan Hutchinson, were general surgeons primarily and ophthalmic surgeons in the second place.

Bowman's name is a household word in ophthalmology. Bowman's membrane, his probes, and "stop" needles are known everywhere; but it was his guiding hand which placed ophthalmology as a speciality on a sure footing. Lawrence wrote on syphilis of the eye. Hutchinson's monograph on syphilitic disease of the eye and ear is a classic, and he was the first to describe the notched incisors in congenital syphilis which will always be known as "Hutchinson's teeth." It is reputed that a French surgeon who visited Moorfields in the early 60's rushed into the out-patient room exclaiming "Where is Monsieur Hutchinson? I want to see his teeth."

Hutchinson's main assistants were Waren Tay and Edward Nettleship. Tay never cared for publicity and was glad to remain in the background. Hutchinson's mantle may be said to have fallen on Nettleship, who had a profound influence in the teaching of ophthalmology in his generation.

Argyll Robertson first described the tabetic pupillary reactions which have ever since borne his name; and in an earlier decade Arthur Jacob, of Dublin, first described the layer of rods and cones in the retina in 1819.

One of the first atlases of the fundus was that of Liebreich who was ophthalmic surgeon to *St. Thomas's* Hospital. He had been turned out of Paris at the beginning of the Franco-Prussian War of 1870, and came to England. There have been several good atlases since his day, notably that of Frost, the pictures in which are still unsurpassed.

Ophthalmic pathology has been well served by the long line of curators of the museum at *Moorfields* Hospital. Nettleship, Lawford, Treacher Collins, all made notable contributions in this subject, but George Coats, who died in 1915, probably contributed more papers of lasting value than any other, with the exception of Sir John Parsons whose magnificent *Pathology of the Eye* in four volumes has long been a credit to British ophthalmology.

For many years the importance of the state of the refraction of the eye was overlooked and underestimated. A case of Brudenell Carter's, published in the Clinical Society's *Transactions*, in 1875, was one of the first to call attention to the importance of this branch of work; and partly in consequence a demand arose for hand-books on refraction; among these, that of Hartridge ran through many editions and was deservedly popular.

In the field of medical ophthalmology, the manual by Sir William Gowers was pre-eminent for many years: its third edition was brought out by Marcus Gunn who, from his experience as Ophthalmic Surgeon to the *National* Hospital, Queen Square, did much to advance ophthalmology in its relations to neurology. Perimeters have been invented by McHardy, by Priestley Smith and by Sir William Lister. Priestley Smith won the Jacksonian prize at the *Royal College of Surgeons* in 1878 with his essay on glaucoma. Few men have had more influence on their speciality of recent years than this celebrated Birmingham ophthalmologist.

Ophthalmological Education

Postgraduate education in ophthalmology is given at the universities and at all the great eye hospitals in London and the provinces. Courses of lectures which include all ancillary subjects such as optics, operative surgery, bacteriology and pathology are given during the terms, and at the same time the student takes his place as clinical assistant to one or more of the members of the senior staff in the out-patient department. If time is no objection, he is wise to hold the post of House Surgeon for a year or more at one of the Eye Hospitals. Even if he does not, he obtains a thorough grounding in clinical ophthalmology, and it is this aspect of teaching which has produced so many of the great names of British ophthalmology during the past hundred years. Special diplomas notably the D.O. Oxon. (1907) and the D.O.M.S. (1920) are awarded to students who pass the qualifying examinations,

and for many years ophthalmology has been one of a number of special subjects which can be taken in the examination of F.R.C.S. (Edinburgh).

Preventive and Social Aspects of Ophthalmology

Steady progress has been made during the present century both in prevention of blindness and in amelioration of the conditions of the blind. Blind pensions are now awarded, and ophthalmic monetary benefits under the National Health Insurance came into force in 1925.

Early in its history the *Ophthalmological Society* appointed a deputation, led by Bowman and Hutchinson, to interview the Home Secretary and draw attention to the ravages caused by *ophthalmia neonatorum*. It was pointed out that this affection is largely preventable, and it was suggested that the Registrar of Births should hand each person registering a birth a printed paper telling him what to do if the baby's eyes showed any signs of inflammation. No action was taken and little was done by the authorities until the *Metropolitan Asylums Board* established *St. Margaret's* Hospital in 1918 for the treatment of *ophthalmia neonatorum*, affording facilities for the admission to hospital of both mother and child. The *Board* also did good pioneer work by establishing, in 1903, residential schools for the treatment of trachoma in children, at the same time ensuring the continuance of their education.

The *London County Council*, at the instigation of Mr. James Kerr and Mr. Bishop Harman, made a notable advance by segregating partially-sighted children in special—so-called "myope"—schools. Valuable reports on the prevention of blindness have emanated from Glasgow in 1926 and in 1942, and from the Union of Counties Association for the Blind in 1932; and the Ministry of Health has at last set up a Standing Committee on the Prevention of Blindness.

REFERENCES

James, R. R. (1933), *Studies in the History of Ophthalmology in England prior to 1800*, Cambridge

Sorsby, A. (1933), *A Short History of Ophthalmology*, London

SPECIAL CONTRIBUTION

THE TASK OF OPHTHALMOLOGICAL RESEARCH

by ARNOLD SORSBY, M.D., F.R.C.S.

Research Professor in Ophthalmology, Royal College of Surgeons and Royal Eye Hospital

I. Blindness: The Extent of the Problem

The Census returns in Great Britain, like those of most countries, used to show the number of blind persons (as also the number of those afflicted with certain other physical disabilities). These returns, based on information supplied by the persons affected, or by relatives on their behalf, had the limitations inherent in any lay assessment of a technical problem, and the number of 26,366 persons returned as blind in the 1911 Census of England and Wales must therefore be taken with some reserve. With the Blind Persons Act of 1920, the practice of seeking to assess the number of blind by Census enquiries was discontinued as it was believed that the Register of those who would seek the statutory benefits (blind pensions, training facilities, etc.) which the Act allowed would be a more complete representation of the position, particularly as only those certified blind by a medical examiner would be registered.

The first year of registration (1919) showed the number of blind as 25,840, a close approximation to the Census figure, but this number has increased steadily year by year. It was 30,785 in 1920, 34,894 in 1921 and reached over 80,000 in 1942. It is likely that even now not all the blind are registered and that a figure of about 100,000 blind for the population of about forty million of England and Wales is not an under-estimate. This gives a rate of blindness of 2·5 per 1,000, the criterion of blindness being "so blind as to be unable to perform any work for which eye-sight is essential."

On the basis of Census returns of various countries it was estimated in 1910 that there were about 3,000,000 totally blind throughout the world, but double that number has been suggested as nearer the truth. If the rate obtained for England and Wales is taken as an index for the world population, 5,000,000 would represent the blind throughout the world. But this figure is an obvious under-estimate, as the rate for blindness in countries like India and China, and throughout the Middle East, is known to be very much higher than that of England and Wales. China alone has probably about four to five million blind, and the number in India cannot be much less. Although nothing approaching an accurate estimate is possible, it is likely that there are, throughout the world, about 15,000,000 blind and many more millions with grossly defective sight.

As distinct from the immediate exciting causes of disease the great determining factors of blindness are ignorance arising from low cultural levels in communities and lack of facilities due to poorly developed medical and social services. It is therefore natural that the incidence of blindness should vary enormously in different countries as these factors are extremely variable. Within a homogeneous community economic status is a powerful determinant, as is well shown by an official health survey undertaken by the United States Government in 1936 and covering 2,498,180 persons. It was found that the incidence of blindness amongst the coloured population (mainly negroes) was 146 per 100,000 against 76 for the white population. About 18 % of the surveyed population fell into the "Relief" group, having had to seek relief from public funds at some time during the twelve months preceding the investigation, and the incidence of blindness in this group was distinctly higher than amongst the non-relief group, which in turn gave a differential rate with different income levels per family, as is seen from the following summary table:

	Rate per 100,000	*Ratio to rate in families with income of* $3,000 *and over*
All incomes	83	
Relief	163	668
Non-relief:		
Under $1,000	110	492
$1,000–1,500	59	238
$1,500–2,000	53	192
$2,000–3,000	41	135
$3,000–5,000	27	100
$5,000 and over	33	

II. The Prevention of Blindness

(i) *Infective Processes*

Whether ophthalmology has shared in the general advance towards the control and eradication of infection as a factor in disease is a debatable and not altogether academic point. It is true that prevention has all but abolished *ophthalmia neonatorum* as a cause of gross visual defect and blindness, and that affections like small-pox are no longer a significant cause of eye disease. Nevertheless, the outstanding ocular infections—trachoma and the acute ophthalmias of tropical countries—are not under control. In the case of trachoma—which affects probably about 15 % of the world's population—the exciting cause is not known with any certainty, and the acute ophthalmias of tropical countries, with their devastating lesions, are a reflection of immensely difficult problems of general sanitation and standards of living. In more developed countries, tuberculous sensitization is responsible for phlyctenular ophthalmia, the prevalent ophthal-

mia of childhood, and here the control of tuberculosis has given gratifying results in the steady decline in the incidence of the affection. Ophthalmology has undoubtedly benefited from the advances in public health and hygiene; it has also helped to limit the local infectious diseases of the eye, but it has done so more by the application of general health measures than by the specific eradication of infectious lesions.

The acute ophthalmias, tuberculous and syphilitic lesions of the eye, and trachoma exemplify the problems of ocular infectious disease. The acute ophthalmias, which are caused by a variety of organisms such as the Koch-Weeks bacillus (*Haemophilus influenzae*), the Morax-Axenfeld bacillus and the gonococcus, rank before trachoma as a cause of blindness in tropical and sub-tropical countries; the causative organisms are known, though our knowledge of the spread of these organisms is still fragmentary. It is clear that lack of cleanliness, congestion and poor sanitary conditions favour the spread of such infections, but the role of climatic, meteorological and possibly ecological factors has still to be assessed. Complex problems of individual immunity also await solution, but it is gratifying that in the meantime the sulphonamides are giving excellent results in what is still a terrible affection.

As in the acute ophthalmias, there is no mystery as to the exciting cause in tuberculous and syphilitic eye disease, but the problem here—in view of the rarity of primary ocular tuberculosis and syphilis—is how to overcome the local effect of a widespread endogenous infection. In tuberculosis and tuberculous allergy of the eye the absence of any specific treatment for tuberculosis makes the ophthalmic condition part of the general problem. In ocular syphilis a special problem is involved as the blood-eye barrier renders specific anti-syphilitic treatment largely ineffective. An anti-syphilitic agent that would reach and act on the brain and eye would be of immense value, for syphilis is responsible for about 20% of all blindness in the more advanced countries.

The difficulties of virus disease are well exemplified by trachoma. Isolation and culture of the infecting agent and the development of a specific therapy, are all as yet unsolved problems. The value of the sulphonamides in trachoma, though these are far from constituting a specific therapy, makes it possible to hope that, as in other affections, an effective remedy may be at hand even if the actual discovery of the mechanism of the disease has yet to come.

(ii) *Genetic Affections*

As infection diminishes in significance as a factor in ocular pathology, genetic disturbances assume an increasingly important position. Before the introduction (1881–84) of the Credé method [local cleansing and instillation of 1–2% silver nitrate] *ophthalmia neonatorum* was responsible for about 40% of blindness in blind schools. Now some 50% of blind children are blind from malformations and hereditary anomalies, as is seen from such studies as those conducted by Berens, Kerby & McKay (1935), and other observers. At present the possible lines on which promising work can be done are still obscure, but the emergent tendency not to make a rigid distinction between genetic and environmental factors is hopeful. Genetic disease is no longer regarded as a fixed entity. It is known that environmental factors can excite or mask an inherent genetic tendency. The control of a genetic affection such as acholuric jaundice by splenectomy, or of Niemann-Pick disease by controlled fat intake, points to the possibility of repeating clinically what can be achieved in the laboratory. The basis for *retinitis pigmentosa* and other abiotrophies is undoubtedly chromosomal, but the mechanism whereby they become manifest may well be somatic.

The limitations and possibilities of genetic research are well shown by *glioma retinae*. The recognition that this is a dominant hereditary disease in some families, places in question the status of the more common (though still rare) sporadic forms. The fact that the mode of inheritance is an irregular dominant, focuses attention on the unaffected transmitters of the disease. What are the genetic or environmental factors that lead to the suppression of the genetic potentiality towards glioma in such individuals?

(iii) *Nutritional and Metabolic disorders*

Nutritional disturbances leading to severe ocular lesions are exemplified in an extreme form by xerophthalmia, which occurs sporadically in Europe but is a major problem in countries like China, India and others of the same economic level. Less severe manifestations are a variety of ill-defined lesions due to various dietetic deficiencies. The correlation of nutrition with ocular health is still largely an unexplored chapter.

Equally obscure are the lesions of faulty general and local metabolism. Characteristic of this obscurity is the fact that the basis of diabetic retinitis is still undetermined. One difficulty underlying such investigations is the absence of any method of studying the pathological physiology of the eye. It is unlikely that an understanding of diabetic retinitis will be reached from the study of the fixed retina under the microscope, or from the biochemical analysis of the blood. Suitable methods for investigating the respiration and total metabolic activity of the intact eye will no doubt be evolved.

(iv) *Cataract and Glaucoma*

Both English and North American statistics on blindness show cataract and glaucoma to be responsible for 25–40% of all blindness. Even in countries ravaged by the acute ophthalmias and trachoma, they are formidable factors, and are naturally more significant in the higher age-groups. It is fortunate that the treatment of these two affections is in advance of knowledge of their pathology, but a fuller knowledge is essential if much blindness, and still more gross visual defects, are to be avoided. The problem here, as in the metabolic disorders, is the evolving of methods of study of the normal and pathological physiology of the eye. Analyses of opaque lenses, valuable as such procedures are, do not give much information on the processes that lead to opacification, and an exclusive physical or chemical approach to glaucoma is an over-simplification of a complex dynamic process.

(v) *Injuries*

Injuries rank high as a cause of the loss of one eye, especially in children and young adults, and are a considerable factor in the incidence of total blindness. In available English statistics, they are not so significant as in American statistics, where an incidence of 15% is often reached. Although blindness from casual injury has tended to decline in importance owing to more adequate treatment and the realization of the significance of sympathetic ophthalmia, blindness from injury as a whole tends to increase for a number of reasons. Traffic accidents in power transport, the expansion of heavy industries and of the chemical industry—to say nothing of the liberal use of high explosives in total war—all contribute to this increase. The wars of the past 70 years have been marked by an ever-increasing rate of eye injuries in relation to the total of wounds, and the present war has brought the difficult problem of extracting non-magnetic fragments from the interior of the eye, as present-day missiles are largely non-magnetic in contrast to the missiles used in the 1914–18 war. Whilst the abolition of war injuries presents a formidable political and social problem the prevention of industrial eye injuries is a tangible possibility.

War is the greatest single cause of blindness, as Kay Sharp, the Chairman of the Prevention of Blindness Committee, has pointed out. As an aftermath of the present struggle there will inevitably be starving populations, disorganised health and medical services, and possibly widespread epidemics carried by moving populations. All these are powerful determinants of eye disease, and ophthalmology will have to evolve not only services for succour, but research facilities to study a whole set of new conditions.

III. Ocular Hygiene

A hundred years ago "asthenopia" was regarded as the first stage of "amaurosis." The clear appreciation of the refraction and accommodation of the eye made possible by the work of Donders (1863) has led to an immense increase in comfort in the large sections of the population suffering from refractive anomalies—a matter of considerable importance in an industrial and educational age. It is difficult for a generation which takes optical aids as a matter of course to realize what a great handicap the absence of such aids constituted to their predecessors. So great is their value that the conditions under which the patient works have tended to be ignored. It is good that the investigation of refractive errors and muscle anomalies is constantly undergoing additional refinements, the latest example being the studies of Ames and his collaborators on optical imagery. But the need for detailed studies of conditions under which work is being done is equally great. To study the patient (or his eyes) only

and to ignore his environment is a one-sided approach. A great deal of industrial injury and disease could have been avoided by an earlier appreciation of this truism. Towards the end of the last century much attention was given to the question of satisfactory working conditions for children in a misdirected effort to prevent the development of myopia, but this has been of great value in laying the basis, not indeed for the prevention of myopia, but for the evolution of the concept of ocular hygiene. Intensive studies on environmental conditions—such as lighting and rest pauses—are needed to supplement the studies in ocular physiology.

IV. The Organisation of Ophthalmology

The rapid expansion of ophthalmic facilities in most countries has tended to be practical rather than academic. There was so much to be done in applying the great discoveries of the nineteenth century to daily life, that the more academic aspects have suffered. Nevertheless the future of ophthalmology, as of all clinical studies, depends on constant invigoration from new investigations dealing with the changed conditions and expanding the older knowledge. Ophthalmological research as a career is an essential need in the reorganisation of ophthalmic practice that is proceeding to-day. In England there is the prospect of creating a well-organised national eye service which will bring the benefit of specialised knowledge, under satisfactory conditions, to the whole population. In such a re-organised service, ample provision should be made for ophthalmic research institutes, where problems can be studied away from the pressure of the clinic, and where ophthalmic needs can be visualized from a better vantage point than the crowded out-patients' department.

The author of this article is the first holder of the Research Professorship in Ophthalmology which was established in 1943 *jointly by the Royal College of Surgeons and the Royal Eye Hospital, London.*

The growing recognition in Britain of the need for ophthalmological research has also been reflected by the formation of the University of Oxford Ophthalmological Research Endowment Committee, which has recently sponsored an appeal for funds for the endowment of a Department of Ophthalmology at Oxford for research, teaching and treatment.

Professor Sorsby, who holds the posts of Surgeon and Director of the Research Unit at the Royal Eye Hospital, was formerly the Dean of its Medical School. He is also Consulting Ophthalmologist to the London County Council, co-editor with Mr. F. Ridley of "Modern Trends in Ophthalmology" (1940), *and author of "A Short History of Ophthalmology"* (1933) *and a more general work "Medicine and Mankind"* (1941).

OPHTHALMOLOGY

161

A STATISTICAL ANALYSIS OF 3,219 PERSONS CERTIFIED BLIND AT THE REGIONAL CLINIC FOR CERTIFICATION OF THE BLIND, GLASGOW AND SOUTH-WEST SCOTLAND, DURING THE PERIOD 1929–1935

by J. Marshall & H. E. Seiler, *British Journal of Ophthalmology*, **26**, 327–380, 385–414 & 433–466, August, September & October 1942

In 1929 a clinic was set up in Glasgow for applicants for certification as blind persons eligible for certain statutory benefits. This clinic dealing with people from Glasgow and South-West Scotland ensured a uniform method of certification, and it was found that 3,216 (62·6%) out of 5,140 applicants during the period 1929–35 were blind within the meaning of the Blind Persons Act ("so blind as to be unable to perform any work for which eyesight is essential"). A detailed analysis of the findings in these 3,216 persons is given in the present paper and may be taken as representative of the causal factors of blindness in Great Britain, particularly as great care was taken both in the examination of applicants and in the statistical evaluation of the findings.

The three outstanding causes of blindness were cataract, myopia and venereal disease, with an incidence of 16·8%, 16·3% and 13·95% respectively. Chronic infective states (chronic septicæmia, autointoxication, *etc.*) accounted for 10·7%, and glaucoma for 8·7%. The incidence for congenital (developmental) anomalies and heredo-degenerative lesions was 7·45 %, whilst injury as a cause of blindness came surprisingly low with 6·4%, though it rose to 10·1% if the cause of blindness of one eye only was considered in patients with bilateral blindness due to two different causes.

Calculations based on the duration in years of loss of sight for each cause of blindness and the number of cases, showed that some of the major causes of blindness were not responsible for the same amount of disablement as that produced by other numerically less frequent causes. Cataract, for instance, fell from the first place to the eighth on the basis of this computation, whilst injury rose from the eighth place to the third, and congenital anomalies from the seventh to the second. On this basis chronic septicæmia took the first place with 12·0%, myopia came fourth with 10·3% and ophthalmia neonatorum fifth with 10·1%. Congenital syphilis was responsible for 7·1% and acquired syphilis for 3·8%.

The age distribution of the different causes of blindness revealed the following points of interest. In the first four years of life ophthalmia neonatorum and congenital anomalies were responsible in 68% of cases of blindness. In the school period (5–15 years) congenital syphilis and non-industrial eye injury were the major causes. In the age-group 16–29 years, syphilis was the most important single cause, but other important causes were injuries (17·3%), chronic septicæmia (16·3%) and myopia (13·3%). In the age-group 30–49 years syphilis, chiefly acquired syphilis, was responsible for one-fifth of the cases, with myopia next in importance with 17·9%. In the period 50–59 years inflammatory conditions gave way to degenerative lesions: myopia and cataract were responsible for 22·7% and 22·1% respectively. In the age-group over 70 years, cataract and glaucoma were the cause of 67% of blindness.

As to sex distribution, diabetes as a cause of blindness showed a significant excess in women, whilst men showed a considerably higher incidence of blindness due to heredo-degenerative lesions, glaucoma, acquired syphilis, injury, sympathetic ophthalmia and vascular lesions.

Amongst other points discussed in this exhaustive study are occupation as a factor in the development of blindness and the different types of complications seen in myopia at different age-periods.

162

ON SOME ANOMALOUS FORMS OF AMAUROTIC IDIOCY AND THEIR BEARING ON THE RELATIONSHIP OF THE VARIOUS TYPES

by R. Wyburn-Mason, *British Journal of Ophthalmology*, **27**, 145–173 & 193–207, April & May 1943

Since the clinical description by Tay in 1881 and the pathological account by Sachs in 1887 of the affection now known as Tay-Sachs disease, a whole series of somewhat similar clinical manifestations have been recorded. The unifying features of these affections are predilection for cerebrum and retina, the broadly similar histological features, and the relentless clinical course. The designation cerebro-macular disease, with such qualifying terms as infantile, juvenile, late infantile, late juvenile and adult types, crystallizes this unifying view. None the less there are valid reasons for doubting the accuracy of this teaching. Clinically each of these different "subdivisions" of "cerebro-macular disease" is so clearly distinct, and the genetic behaviour so markedly different, that it is difficult to visualize these different features as one. Moreover, the histological resemblances are by no means so definite as earlier observers believed; the chemical composition of the degenerate nervous tissue is distinctly different in these different affections; and it is also of considerable significance that there is no substantial

evidence of the occurrence of the different manifestations of this alleged unitary affection within the same family.

A useful contribution to this complex problem is made by the author of the present paper. In addition to reporting a number of fairly "typical" examples of the different forms of cerebro-macular disease, he describes the case of a girl aged 9 years and 9 months, suffering from indefinite neurological symptoms and showing the "cherry-red spot" characteristic of Tay-Sachs disease. This well-authenticated observation raises the question as to the validity of regarding the "cherry-red spot" as pathognomonic of the infantile form of cerebro-macular disease. In this case there was some indefinite evidence of some generalised anomaly of the lipoid metabolism, and this finding only accentuates the problem raised by previous writers as to whether Tay-Sachs disease is itself at least two distinct entities: one being a neurological degeneration and the other a generalised lipoid disturbance—the Niemann Pick disease—with fatty infiltration of the cerebrum and retinæ. It is unlikely that the prevailing confusion will find an easy solution, but observations like the one under discussion help in the elucidation of a distinctly obscure chapter in neurology and metabolic disorders.

163

THE ÆTIOLOGY OF PHLYCTENULAR OPHTHALMIA

by A. Sorsby, *British Journal of Ophthalmology*, **26**, 159–179 & 189–215, April & May 1942

In Great Britain, as elsewhere in Europe and in the United States, phlyctenular ophthalmia is much less frequent than it used to be. The decline in incidence has continued steadily throughout the present century except for the years of the 1914–18 war and its aftermath. Towards the end of the last century many different bacteria had been incriminated as the cause of the affection, but evidence that the condition is somehow associated with tuberculosis emerged at the beginning of this century, when many observers found a high incidence of positive tuberculin reactions in children affected with phlyctenular ophthalmia. This evidence was strengthened by the fact that the ophthalmo-reaction to tuberculin, used as an alternative to the skin reaction, did in itself produce phlyctenular ophthalmia in some children. Further support for the tuberculous nature of the affection came with the introduction of chest radiography, when it was found that a high percentage of affected children showed some evidence of tuberculosis of the lungs.

Nevertheless, the tuberculous nature of phlyctenular ophthalmia could not be accepted as validly established, and this was for two reasons. In the first place the tubercle bacillus could not be found in the phlycten, and secondly the affection was but rarely associated with manifestations of clinical tuberculosis; phlyctenulæ were not often seen in frank cases of pulmonary or abdominal tuberculosis, nor could such generalised lesions frequently be found in children suffering from phlyctenular ophthalmia. How these contradictory data have come to be harmonised is shown in the present exhaustive study on the ætiology of phlyctenular ophthalmia. The author has had the advantage of studying the problem in a country (England) where tuberculosis is not common, and his findings are therefore free from the possible fallacy that any evidence of tuberculosis he observed in his cases is coincidental. Using the unique material available at *White Oak* Hospital, near London, where special facilities exist for children with phlyctenular ophthalmia and other chronic eye affections, the author found positive tuberculin reactions in 84·8% of 592 children under 16 years old suffering from phlyctenular ophthalmia against 15·3% in a control group of 900 cases of blepharitis. Radiological evidence of tuberculosis was present in 72·2% in the phlycten cases and in not more than 16·1% in the controls. Evidence of clinical tuberculosis was found in 6·4% of the phlycten cases, against not a single case in the control series. A family history of tuberculosis was present in 28·9% of the phlycten cases against a computed incidence of 3·7% for the normal school child population of London.

Of particular interest was the after-history of 754 phlycten cases treated at the hospital during 1921–31; 5·3% of these showed a subsequent incidence of clinical tuberculosis, and a tuberculosis mortality rate of 0·8%, in contrast to a morbidity rate of 0·8% and a mortality rate of 0·1% for 1,024 controls. Of significance, too, are the author's negative findings. Tubercle bacilli could not be recovered from gastric lavage of 50 cases, nor in the excised tonsils of 20 others. Tests for bacterial allergy, other than tubercular, were negative, and there was no evidence that malnutrition, focal sepsis or pediculosis are seen more frequently in phlyctenular ophthalmia than in control cases of blepharitis.

The author concludes that the evidence shows tuberculous infection, rather than tuberculous disease, to be the underlying factor in phlyctenular ophthalmia. He suggests that the phlycten appears only when a hyperallergic phase of this infection is present and there is a suitable (? specific) exciting factor of endogenous or exogenous origin, such as metabolites, tubercle bacilli or tuberculous dust. In fact, the phlycten may be described as a spontaneous tuberculin test reaction, but its significance is more grave than that of an artificially induced positive tuberculin reaction in a child: this is borne out by the subsequent incidence of tuberculosis and mortality from it in children previously affected with phlyctenular ophthalmia. The gratifying fact is that most children with tuberculous infection and phlyctenular ophthalmia overcome the infection and do not develop clinical tuberculosis.

164

THE PRODUCTION AND TREATMENT OF EXPERIMENTAL HYPOPYON ULCERS IN THE RABBIT

by J. M. Robson & G. I. Scott, *British Journal of Experimental Pathology*, **24**, 50–56, April 1943

Continuing their studies (Robson & Scott, 1942; 1943) in local chemotherapy on experimental corneal lesions in the rabbit, the authors report, from the Department of Pharmacology, Edinburgh, an investigation of the value of sodium sulphacetamide, sodium sulphapyridine, penicillin and tyrothricin on artificially induced pneumococcal infections. By injecting into the cornea pneumococcus type 19 (a 6 to 15 hours' culture in plain broth) they succeeded in obtaining clearly defined corneal ulcers frequently associated with hypopyon. Definite beneficial effects were obtained with penicillin and 30% sodium sulphacetamide, but especially with the first. Tyrothricin was of little value, and 30% sodium sulphapyridine tended to produce secondary effects by the deposition of the powder. In the use of sodium sulphacetamide, delay for more than 6 hours greatly decreased its value; with penicillin, quite appreciable effects were still obtained when treatment was delayed for 24 hours.

REFERENCES

[1] Robson, J. M., & Scott, G. I. (1942), *Brit. med. J.*, **1**, 5

[2] Robson, J. M., & Scott, G. I. (1943), *Lancet*, **1**, 100

[1] [*typescript abstract available on request*]

[2] [*see BMB* **53**]

165

THE SULPHONAMIDES IN EXPERIMENTAL OCULAR INFECTIONS

by M. Klein & A Sorsby, *British Journal of Ophthalmology*, **27**, 241–254, June 1943

The authors, the second of whom has recently been appointed Research Professor of Ophthalmology at the *Royal College of Surgeons*, give a general survey of the position of both general and local chemotherapy in experimental ocular infections and report a series of experiments which substantiate the value of the sulphonamides for general administration in streptococcal and possibly pneumococcal experimental infections of the interior of the eye. They could not substantiate the value of the sulphonamides for local administration in either streptococcal or *B. pyocyaneus* infections and could not obtain any evidence of the efficacy of the sulphonamides against corneal lesions from *B. pyocyaneus* even when the drugs were given subcutaneously. They stress the need for a more standardised technique of inducing experimental infection of the cornea with the organisms commonly seen, and maintain that the problem of the relative value of the general and local use of the sulphonamides still remains unsolved, and that the value of local therapy is still an open question.

166

AVASCULAR HEALING IN THE CORNEA

by B. D. Pullinger & I. Mann, *Journal of Pathology and Bacteriology*, **55**, 151–158, April 1943

In this paper from the laboratories of the *Imperial Cancer Research Fund*, London, and the *Nuffield* Laboratory of Ophthalmology, Oxford, the authors report a study of the healing of experimental lesions with liquid dichlorodiethyl-sulphide (mustard gas) in the eyes of rabbits. The same authors (1942) have previously reported on the clinical pathology of such experimental lesions. Spontaneous avascular healing invariably followed when the applications of the liquid droplet damaged the centre of the cornea alone, leaving the corneo-scleral junction uninjured and free from oedema. That healing occurred by sliding down of limbal epithelium was readily observed macroscopically when rabbits which were pigmented at the corneo-scleral junction were used. In the course of healing, the substantia propria was invaded by large numbers of wandering cells, readily demonstrated by the use of vital staining. Some of these cells acted as macrophages, others were transformed into keratoblasts (corneal fibre-forming cells) and fibrocytes, while some probably became corneal corpuscles. The original paper is illustrated by 13 photomicrographs.

REFERENCE

Mann, I., & Pullinger, B. D. (1942), *Proc. roy. Soc. Med.*, **35**, 229

167

WAR SURGERY OF THE EYE: Removal of Magnetic Intra-ocular Foreign Bodies by the Posterior Route

by H. B. Stallard, *British Medical Journal*, **2**, 629–631, 28/11/42

The outstanding problems in the removal of intra-ocular foreign bodies are localisation and method of operative approach. Under war conditions a simple technique of localisation is necessary and the present author advocates a radiological procedure suggested by Brigadier C. W. Graham, consulting ophthalmologist to the British Army in the Middle East.

The principal points of this technique are as follows: The eye is anæsthetised with cocaine, and when it is much inflamed 2–3 minims [0·12–0·2 cm.³] of a procaine solution are injected into the episcleral tissues at the limbus at the 12, 9, and 3 o'clock positions. A silver ring of a size to fit exactly the corneo-scleral junction is stitched in position by sutures of 000 gauge silk passing through the conjunctiva at 12, 9, and 3 o'clock. A drop of liquid paraffin is instilled into the eye, and a pad moistened in the oil is applied to the closed lids and bandaged in position. When possible the patient sits during *x*-ray examination, but in some cases other wounds prevent this. He is directed to look forwards during the first exposure and downwards for the second. For this purpose conspicuous marks such as red disks or lights 5 cm. in diameter are placed on the wall for sitting cases, and on the ceiling for lying cases, at the two points at which fixation of the eyes is necessary during exposure. Each exposure is half the normal. To obtain a postero-anterior view of the orbit free from the dense shadow of the petrous part of the temporal bone the head is tilted slightly so that the occiput is down and the face up; the petrous shadow then falls over the antra. In an accurate postero-anterior radiograph the silver ring shows as a perfect circle, and in the lateral radiograph as a linear shadow.

The interpretation of these radiographs offers no special difficulties. Movement of the foreign body is the essential diagnostic feature. If the relation of the silver ring image to the foreign body is unaltered in the first and second position it may be presumed that the foreign body is moving with the eye. The radiographs with the schematic eye marked upon them will indicate also the position of the object with regard to the centre of rotation. If the foreign body is in front of the centre of rotation it will apparently move with the ring; if it is behind it will move in an opposite direction—that is, a foreign body in the posterior half of the eye will lie at a higher level in the second position where the ring has rotated downwards.

The shadow of the foreign body might fall within the circles described and yet be outside the eye. This point is settled by the movement of the foreign body, which is absent when it is extra-ocular. There is, however, an exception to this in the case of a foreign body in Tenon's capsule, in which case slight movement may take place, but this is rarely as great as in the case of an intra-ocular foreign body.

For removal of foreign bodies the author advocates the posterior route. This has the advantage of avoiding injury to the ciliary body, iris and lens, and is of particular value for non-magnetic foreign bodies—a point of considerable importance, as many foreign bodies arising from missiles used in the present war are non-magnetic. In the author's series the foreign body was visible with the ophthalmoscope in 50 per cent. of cases; in the remainder it was obscured by vitreous hæmorrhage. In such patients localisation must be by radiography. By the use of surface diathermy, 70 to 80 mA for 5 seconds, and a mattress suture (000 silk threaded on a Merson small eyeless needle) passed through half the thickness of the sclera, control of bleeding and controlled exposure of the field of operation is readily obtained. Incision of the sclera and instrumental removal of the foreign body can then be carried out. Operative results by the technique described have been good.

168

VITAMIN A AND DARK ADAPTATION

by J. Yudkin, G. W. Robertson & S. Yudkin, *Lancet*, **2**, 10–13, 3/7/43

In this paper from the *Dunn* Nutritional Laboratory (Cambridge) and the Department of Chemical Pathology, *University College* (London), the authors describe the technique employed in testing the dark-adaptation of 400 people. The apparatus used was that described by Haines (1938). The subject looks down a tube 14 inches [about 35 cm.] long which is whitened on the inside and is illuminated by a 75-watt lamp. The test is conducted by switching on the bright light and instructing the subject to rest his head against the end of the tube and look along it. After seven minutes the bright light is replaced by a test light and a white plate bearing black arrows pointing in different directions is rotated into place so that one of the arrows is visible. It takes the subject about 20 to 30 seconds to see the arrow, then the intensity of the illumination is still further decreased. This procedure is repeated until 30–40 minutes have elapsed, when dark-adaptation is usually complete. After 30 minutes of adaptation, the threshold is usually determined by a procedure which is the reverse of that used in the earlier part of the test. The minimum detectable illumination is determined by increasing the light until the arrow is just seen and then slowly reducing it until it disappears. This is repeated about three times, after 30 minutes, after 35 minutes and after 40 minutes of adaptation.

Using this technique the authors found that they were able to get results which could be repeated, within narrow limits, for any one individual. They found also that the visual threshold, particularly in the first ten minutes, did not necessarily bear any relation to the final rod-threshold. This observation is important because many people still attempt to assess dark-adaptation by methods which give readings within a few minutes or seconds of the onset of dark-adaptation.

The four possible ways in which vitamin A may affect dark-adaptation are by alteration in:

(i) Final rod threshold only,
(ii) final rod threshold and final cone threshold,
(iii) final rod threshold and cone-rod transition time,
(iv) final rod threshold, final cone threshold, and cone-rod transition time.

In an intensive study on 14 subjects of the effect of vitamin A (24,000 international units daily) and carotene (20,000 international units daily) on dark-adaptation, the authors observed each of the four types of alteration, and these are depicted in the original paper by a series of curves.

When vitamin A has any effect at all it always affects the final rod-threshold. The cone-threshold or the rod-cone transition time may or may not be affected.

One of the main conclusions from these findings is that techniques of assessment of visual performance in the dark must take into account the requirements of the subject.

For those engaged in outdoor night work for long periods,

the final rod threshold is probably of most importance, whereas the cone-rod transition time is of special importance for those who must alternate rapidly between dark surroundings and bright light.

REFERENCE
Haines, R. T. M. (1938), *Trans. Ophthal. Soc. U.K.*, **58**, 103

STOMACH IN HEALTH AND DISEASE

169

MOTILITY OF THE FASTING STOMACH IN HEALTH AND DISEASE

by W. F. Anderson, *Lancet*, **1**, 40–42, 9/1/43

In this paper from the Department of Materia Medica and Therapeutics of Glasgow University the author describes a modification of Carlson's (1916) balloon method for recording the movements of the empty stomach in man. Objections to this technique are discussed and the author concludes that, after an initial disturbance due to inserting the balloon, mechanical stimulation becomes insignificant and the balloon itself does not initiate contractions. Twenty-three healthy subjects were investigated. Three phases in gastric activity were revealed: (i) period of active contractions, (ii) tonus rhythm, and (iii) phase of relative quiescence. A tetanic phase was not observed in the healthy stomach. The oculo-gastric and carotico-gastric reflexes described by Danielopolu (1930) were demonstrated but if the stomach was quiescent the phase of excitability was not elicited in either reflex. The acid content of the gastric secretion varied from complete achlorhydria to well-marked hyperchlorhydria in these subjects, but the duration of the individual gastric contraction remained practically constant, and the amplitude did not appear to be related to the amount of hydrochloric acid in the gastric juice.

In 10 patients with acute duodenal ulcer, gastric tone was increased while individual contractions were stronger than in the healthy stomach. Occasionally a phase of tetanus was demonstrated. In a patient with acute gastric ulcer the contractions were weak, while in one with chronic gastric ulcer they were powerful. In two patients with gastric carcinoma contractions were less frequent and less powerful than in the healthy subject. In several patients with duodenal ulcer the complaint of a spasm of pain coincided with the appearance of a very marked contraction, in one instance tetanus, of the stomach.

The ingestion of cold water by healthy subjects always inhibited gastric contractions, but in 8 patients with peptic ulceration there was either no change or an increase of gastric motility.

REFERENCES
Carlson, A. J. (1916), *Amer. J. Physiol.*, **45**, 81
Danielopolu, D. (1930), *Die viscerographische Methode*, Berlin

170

GASTRIC ACIDITY DURING THE FIRST YEAR OF LIFE

by R. A. Miller, *Archives of Disease in Childhood*, **17**, 198–209, December 1942

Until within recent years it has been generally accepted that gastric acidity was low and increased little throughout the first year of life, and that congenital achlorhydria was probably non-existent. In 1938, however, Cutter noted a rapid rise in the gastric acidity during this period by adopting the use of histamine test-meals. Three years later, Miller (1941) showed that the newborn infant had a very acid fasting juice similar in potency to that of an adult, and that the lowest values in infancy appeared only when the infant was at least two or three days old. Out of the 50 healthy babies thus tested there were 6 exceptions who had achlorhydria from birth.

Further evidence confirming and modifying the above findings is supplied in the present paper from the Department of Child Life and Health of Edinburgh University. The author has demonstrated the development in the digestive power of the infant by examining about 200 test-meals throughout the first year of life. These meals consisted of equal parts of milk and water and amounted to sixty minims per pound [approximately 8 cm³. per kg.] body weight. Three specimens of gastric contents were taken; one after the infant had been starved for 3–8 hours, and the other two ½ and 1 hour after the test-feed had been given. The results of these tests may be briefly stated by saying that the average maximum free acidity was 0 cm³. N/10 free acid and 18 cm³. N/10 total acidity at the end of the first week of life, whereas these values rose to 20 cm³. and 61 cm³. respectively by the end of the first year of life. The incidence of achlorhydria during this period fell from 100 % to 17·5 %.

The author concludes that after an infant is a week old and maternal influence is absent, there is a progressive increase in gastric acidity throughout the first year of life which is accompanied by a decline in the number of achlorhydric infants. These changes are primarily due to the making good of a deficiency in the functional capacity of the stomach, and to a less extent to the actual development of the glands in the gastric mucosa. Consequently congenital achlorhydria, no matter whether true or false, is almost always a transitory and not a permanent phenomenon during the first year of life. In addition, it was noted in two cases of pyloric stenosis that gastric stasis and abnormalities in the gastric acidity occurred only after the onset of vomiting. Similarly, in seven babies who had suffered from eczema the test-meal results did not differ appreciably from the normal in the early stage of the disease. It is concluded that any change observed in these diseases is not primary but secondary.

REFERENCES
Cutter, R. D. (1938), *J. Pædiat.*, **12**, 1
Miller, R. A. (1941), *Arch. Dis. Childh.*, **16**, 22 & 113

171

EVALUATION OF GASTROSCOPY

by A. Morton Gill, *Lancet*, **1**, 333–334, 13/3/43

The author, from experience gained in an *Emergency Medical Service* Gastro-intestinal Unit [described by Gill, Berridge & Jones, 1942], suggests that a soldier with dyspepsia profits from complete investigation, including gastroscopy, and that it does not aggravate his symptoms. The soldier accepts the results, and further investigation is rarely required or justified. In order to substantiate this latter claim the results of 1,000 consecutive gastroscopies on service cases are analysed in the present paper.

Analysis of Material. The cases have been divided into 3 groups, A, B, and C, in accordance with the radiological findings.

Table of Gastroscopic Findings

	Whole series		*Group*		
	No.	%	A	B	C
Normals	295	36·6	60	164	71
Ulcer, including benign pyloric stenosis	43	5·3	34	9	0
Gastritis:	396	49·1	201	102	93
Superficial	*196*		*89*	*63*	*44*
Hypertrophic	*166*		*98*	*21*	*47*
Atrophic	*34*		*14*	*18*	*2*
Postoperative	6	0·7	6		
Tumours	4	0·5	2	1	1
Miscellaneous	51	6·3	19	22	10
(the largest group here is multiple erosions)	*36*		*16*	*15*	*5*
Failures	11	1·3	1	10	0
Total (excluding figures in italics)	806		323	308	175

Group A. Both radiography and gastroscopy revealed a gastric lesion. The gastroscope proved of special value here in ulcer cases to demonstrate that final healing had indeed occurred. In the classification of gastritis and the follow-up of the results of treatment, the gastroscope was also of considerable value. The frequency of gastritis will be seen from the table. In general, the superficial and hypertrophic types do not seriously incapacitate soldiers, but the atrophic type, usually a permanent and progressive lesion, requires invaliding. Multiple gastric erosions occurred most frequently in the miscellaneous group, and although they respond well to treat-

ment, they often relapse. Where relapse occurs after treatment, discharge from the Forces is advised. In 60 cases no lesion was seen at gastroscopy, although a positive *x*-ray finding was reported. Fifty-five of these cases were of gastritis, and 5 were of ulcers, usually high in the antral roof.

Group B. No gastric or duodenal lesion was found at *x*-ray. It was concluded that approximately the same number of ulcers invisible to *x*-ray were not seen with the gastroscope. The cases of atrophic gastritis in this group all presented an achlorhydria or a hypochlorhydria. It may be concluded that gastroscopy is indicated in any chronic dyspeptic, or in men with much diminution of the acid secretion.

Group C. No gastric lesion was found at *x*-ray, but abnormality of the duodenum or pyloric canal was demonstrated. There were few important gastric lesions in this group.

Conclusions. A thousand consecutive gastroscopies in military patients lead the author to the following conclusions: In gastric ulcer cases, the gastroscope affords proof of healing, and reveals ulcers not otherwise demonstrable. In chronic gastritis it provides confirmation of the diagnosis, and differentiates its type and severity. Mucosal atrophy can be diagnosed with certainty only with the gastroscope. Multiple gastric erosions are seen well gastroscopically, and differentiated into acute and chronic lesions. Disposal of patients can be arranged accordingly. The Hermon Taylor gastroscope was used throughout.

REFERENCE

[1] Gill, A. M., Berridge, F. R., & Jones, R. A. (1942), *Lancet*, **1**, 727

[1] [*typescript abstract available on request*]

172

POLYPS OF THE STOMACH AND POLYPOID GASTRITIS

by E. Spriggs, *Quarterly Journal of Medicine*, **12**, 1–49, January 1943

During routine examination, with barium *x*-rays, of 4424 persons at the *Ruthin Castle* Clinic, 30 cases of filling defect were seen, due to pear-shaped or nodular masses in the stomach. These were either true polyps, or multiple polypoid outlines due to hypertrophic œdematous folds. The latter condition differs from the usual hypertrophic gastritis by the presence of large nodular masses. The first group totalled 10, or 0·3 %, of all stomachs examined, and an 11th case of polyp in the duodenum was revealed at operation. The second group totalled 19 cases, and 470 patients were found to have gastritis during the same period. The author discusses this series of cases, together with 35 cases of gastric polyps communicated by colleagues, and 54 from the literature.

Pathology. Benign gastric tumours may be papilloma, adenoma, leiomyoma, adenomyoma, neuroma, angioma, lipoma, angio-endothelioma, or cysts. They are usually adenoma or papilloma, leiomyoma, and the polypoid hyperplasia ascribed to chronic irritation.

(i) The papilloma or adenoma may be single or multiple, sessile or stalked. It may be tree-like, and is usually small. The epithelial element may become carcinomatous. This occurs in about 1 in 5–6 cases, considerably less often than in intestinal polyadenoma.

(ii) Leiomyoma is usually single, and may be large. Sarcoma may follow. Ulceration leading to hæmorrhage is not infrequent.

(iii) The hyperplasias are usually the result of a chronic gastritis which at times produces hypertrophic polypoid masses. These appear red and convoluted through the gastroscope.

Polyps : The diagnosis is made radiologically and gastroscopically. The frequency was 0·25 % of patients examined, amounting to 11 cases, and in the same series there were 102 carcinomata, and 5 % of persons had peptic ulcer. The epithelial types of polyp were commonest, followed by the polypoid myomata. The commonest site was the body and pre-pyloric part of the stomach. The average age was 55, and the sex distribution appeared to be equal. About half were single polyps, the rest multiple.

Symptoms in 100 Patients with Gastric Polyps

Gastric pain	51	Weakness	17
Eructation	12	Wasting	17
Vomiting	21	Diarrhœa	8
Nausea	12	Constipation	4
Hæmorrhage and anæmia	33	Anorexia	8
		Palpable tumour	9

Treatment. Where there is hæmorrhage or persistent dyspepsia, or the polyp is large, a wide excision of the tumour and its base is desirable, *i.e.* a partial or subtotal gastrectomy. The patient's condition must of course be suitable, and preoperative treatment including transfusions may be required. Patients with polyps which are causing no symptoms, or symptoms which yield to medical treatment, or patients unfit for operation, have been under observation for years without mishap. Patients with polyps do not usually die of them.

Polypoid Gastritis : The process is not neoplastic, nor does it give rise to tumour formation. In the author's series of 19 cases, there was gastroenterostomy, or past or present peptic ulceration in 7. In 3 of the remaining 12 there was evidence of alcoholic excess. Swallowed septic material was an obvious cause in 1, and a probable cause in 2 others.

Diagnosis is again radiological, and is confirmed with the gastroscope. Some of the masses resemble carcinoma. Clinical features of carcinoma are, however, absent, and no tumour can be felt. Radiologically the flexibility and power of contraction of the stomach are characteristic. Dyspepsia is invariable and has an inconstant food relation. In cases without ulceration, the free HCl was low or absent. In these cases there was degeneration in the gastric epithelium. In 3 patients, progress towards achlorhydria was noted over 2–5 years, and was compatible with clinical improvement.

Treatment. After hæmorrhage, an early gastric ulcer diet is needed, with eggs and milk. Otherwise the amount, nature, and frequency of feeds to be prescribed are individual matters, particularly with achlorhydria. The patient's experience is a valuable guide. Fruit purée and green vegetables are good, and alcohol is best avoided.

Gastric lavage is valuable. A tube may be used, or self-lavage by drinking a pint of water containing a teaspoon of sodium bicarbonate.

The regime prescribed appears to have assisted in promoting regression of symptoms and swellings.

The original 49-page paper contains case-histories, a tabulated summary of the findings in a large number of cases, drawings in colour of gastroscopic and microscopic appearances, radiographs, and drawings and photographs of macroscopic appearances.

173

HÆMATEMESIS AND MELÆNA : With Special Reference to Bleeding Peptic Ulcers

by F. Avery Jones, *British Medical Journal*, **1**, 689–691, 5/6/43

The author, who is physician in charge of the dietetic wards of the *Central Middlesex* County Hospital, reports a full investigation of 171 cases of hæmorrhage from stomach or duodenum. Accurate records were obtained both of the cause of the bleeding and of the results of treatment with large transfusions and early feeding.

It is unsatisfactory to look only at an *x*-ray shadow when the ulcer can be directly inspected through a gastroscope. Gastroscopy was performed on many cases within a week of bleeding. This did no harm, and in 21 cases ulcers were found which had not been diagnosed clinically or radiologically. Consequently a high standard of diagnostic accuracy is claimed for this series. The final diagnoses are shown in the table on p. 109.

Out of these 171 cases there were 17 deaths. This 10 % mortality at first seems high compared with other series such as that of Miller & Nicholson (1941) who reported a 3·1 % mortality in 1396 cases, but allowance must be made for the large number of old people in the present series (30 % were over 60 years of age).

	Admissions			Deaths
	No.	Male	Female	
Chronic lesser curvature ulcer found by *x*-ray, operation, or necropsy	30	26	4	7
Acute gastric ulcer found by early gastroscopy but not visible when radiographed later	21	15	6	0
Acute gastric ulcer found at necropsy	1	0	1	1
Pyloric ulcer	2	2	0	0
Chronic gastric and duodenal ulcers	5	4	1	0
Duodenal ulcer	47	36	11	3
Duodenal ulcer with gastro-enterostomy	6	6	0	0
Jejunal ulcer (gastro-enterostomy)	2	2	0	0
Localized jejunitis	1	1	0	0
Probable peptic ulcer with thoracic stomach	2	0	2	0
Peptic ulcer of Meckel's diverticulum	1	1	0	0
Hæmorrhage complicating perforated gastric or duodenal ulcer	7	6	1	3
Acute gastritis	2	2	0	0
Carcinoma ventriculi	4	2	2	0
Leiomyoma ventriculi	1	1	0	0
Portal thrombosis—gastrostaxis	1	1	0	1
Cirrhosis hepatis	4	1	3	2
Uncertain (6 patients not investigated)	34	21	13	0
Totals	171	127	44	17

Treatment

(*a*) Diet: The patients were given 2-hourly purée feeds, and were allowed to drink an unlimited amount of $\frac{1}{3}$-normal saline between feeds.

Purée Diet

6 a.m.	Cup of milky tea
8 a.m.	Porridge and bemax; thin bread-and-butter and jelly marmalade; cup of milky tea
10 a.m.	Cup of milk and biscuit
12 noon	Minced meat, chicken, or steamed fish; mashed potato; purée carrot or cauliflower
2 p. m.	Egg custard or cereal pudding or apple purée; orange juice
4 p.m.	Cup of milky tea; three slices of thin bread-and-butter; fruit jelly; sponge cake
6 p.m.	Vegetable soup or minced chicken sandwich
8 p.m.	Milk pudding or cup of milk
10 p.m.	Cup of milk and biscuit

Milk feeds during the night if awake

(*b*) Transfusion: Without any rigid rule, blood was usually transfused when the pulse rate remained above 120 per minute, the blood pressure below 90 mm. Hg, or the hæmoglobin below 40%, but any case whom further bleeding might endanger was transfused. With the *Medical Research Council* transfusion apparatus, exposure of the vein was rarely necessary, and both the patients' vessels and the nursing staff were therefore left unperturbed. The blood was given at a rate of 40 drops per minute, or faster in exsanguinated patients. The total amounts usually varied from 1–5 litres. Morphine $\frac{1}{6}$ grain [about 10 mg.] was given when bleeding occurred. Iron and ascorbic acid were given afterwards.

Discussion

In a comparable series of cases treated with *restricted* fluids and *scanty* transfusions (Cullinan & Price, 1932) the death rate was twice that of the present series, and recurrent hæmorrhages more frequent, as is shown in the following table:

	Cullinan & Price	*Present Series (Hæmatemesis cases only)*
Number of cases with hæmatemesis	105	126
Deaths	19	10
Recurrent Bleeding	39	35
Recurrent bleeding and death	16	6

There is thus good evidence favouring the present treatment.

Surgical Treatment

In diagnosed cases only those with chronic gastric ulcers or perforations showed any indications for surgical intervention (which carries a 26% mortality). The dangerous chronic ulcers were those associated with old age, arteriosclerosis, severe pain, or recurrent bleeding, and such cases could possibly be saved by expert surgery. In undiagnosed cases the same indications apply, but the necessity for continued transfusions is no reason for operation.

Organisation

Successful results demand a special diet ward, a staff experienced in this treatment, and an adequate blood bank. Intractable ulcers detected in the follow-up clinic should have partial gastrectomy before complications occur.

REFERENCES

Cullinan, E. R., & Price, R. K. (1932), *St. Bart.'s Hosp. Rep.*, **65**, 185

Miller, T. G., & Nicholson, J. T. L. (1941), *Amer. J. digest. Dis.*, **8**, 446

CARDIOVASCULAR SYSTEM

174

SIGNS SIMULATING THOSE OF MITRAL STENOSIS

by C. Bramwell, *British Heart Journal*, **5**, 24–26, January, 1943

The purpose of this paper from the Cardiographic Department of the *Manchester Royal Infirmary* is to suggest that duplication of the second heart sound at the apex is physiological. Also to put forward a hypothesis to account for this, and for certain other signs that may lead to a mistaken diagnosis of mitral stenosis.

The duplicated second sound is best heard with the patient lying on the left side. This is due to the horizontal and therefore accelerated blood-flow from auricle to ventricle. The second element of the duplication appears to be identical with the physiological third heart sound. Both occur more commonly in young subjects. In 43% of a series of recruits a duplicated second sound was heard, and 70% were fit for service in grade one. Radioscopy usually showed a prominent pulmonary arc. The duplicated second sound was usually associated with a frank apical systolic murmur, or an impure first sound. This murmur may be attributable to a safety-valve mitral incompetence, associated with ventricular over-filling. This is similar to the murmur heard in normal people after severe physical exertion.

Presystolic Murmur. The presence or absence of this murmur is determined by the velocity of the blood-flow through the mitral orifice, not by its size. The author suggests that even with a normal-sized orifice, sufficient increase in the rate of blood-flow will produce a murmur. In the course of a routine examination of 192 long-distance athletes at the Olympic Games it was found (Bramwell & Ellis, 1931) that 12 had a modified first sound indistinguishable from a presystolic murmur. The increased blood-flow may be caused by auricular hypertrophy (part of the general cardiac hypertrophy) in athletes, or in an excitable over-acting heart, as in thyrotoxicosis. In both cases there is a relative mitral stenosis due to increased blood-flow.

Early Diastolic murmur. The classical signs of mitral stenosis are a presystolic murmur, with an accentuated first sound, and a duplicated second sound, in the early stages of the stenosis. Later the duplicated second sound is followed by a diastolic murmur. The second element of the duplicated second sound is due to vibrations of the thickened mitral valve set up by the first rush of blood from the auricle into the ventricle.

In this respect its mode of production is similar to the normal third heart sound. But in the former the sound is attributable to stenosis of the orifice and thickening of the cusps, and in the latter to an increase in rate of blood-flow with a normal valve. The former is the pathological, the latter the physiological, third heart sound.

The author's hypothesis entails a physiological conception of mitral stenosis based on the volume of blood which an orifice of a certain size can transmit in unit time.

REFERENCE

Bramwell, C., & Ellis, R. (1931), *Quart. J. Med.*, **24**, 329

175

CARDIAC SYMPTOMS COMPLICATING DIABETES AND THEIR TREATMENT

by K. S. Smith, *British Heart Journal*, **5**, 1–7, January 1943

The author, who is physician to *Charing Cross* Hospital and the *London Chest* Hospital, records observations on 49 patients

with diabetes and heart disease, discusses the nature of the cardiac syndromes developing, and outlines schemes of treatment.

There is much evidence in the literature to suggest that diabetes is conducive to early and serious heart disease, *e.g.* Root, Bland, Gordon & White (1939), in necropsies on 349 diabetics and 3,400 non-diabetics, found coronary occlusion in 23% of the diabetics and 6% of non-diabetics between 40 and 60 years of age. Other observers have shown general and aortic sclerosis and hypertension to be common in diabetics.

Clinical Observations in the Present Series

The 49 diabetics with heart disease suffered from all grades of diabetes. The heart disease ranged from symptomless hypertension to acute coronary occlusion. Average age was 61 in females and 62 in males and the sex distribution was even. Disease of the heart was always hypertensive or degenerative. 41 cases exhibited hypertension, and the pressure fell considerably under treatment.

Relation of Cardiovascular Disease to Diabetes

Group 1. Patients known to have diabetes and cardiovascular disease : 18 cases. The diabetes is usually of long duration, and coronary thrombosis is common.

Group 2. Patients known to have diabetes, and developing heart disease : 12 cases.

Group 3. Patients known to have heart disease, and developing diabetes : 5 cases.

Group 4. Patients previously believed well, in whom the occurrence of a cardiac or diabetic syndrome brought to light the presence of diabetes or heart disease : 14 cases.

Various Cardiac Syndromes

(i) Effort angina and spasmodic angina : Angina may be of the usual type associated with coronary sclerosis, or it may be related to metabolic disorder of the heart-muscle (in which case improvement may follow insulin treatment).

(ii) Coronary thrombosis and cardiac infarct : The incidence of this is highest amongst diabetics needing, but not taking, insulin, although the latter are not protected from coronary thrombosis. The sudden withdrawal of diet or insulin may precipitate coronary thrombosis.

(iii) Cardiac asthma and congestive heart failure : In diabetics the disturbance of cardiac nutrition may determine the development of failure, and stabilisation may improve the circulation.

Treatment of Cardiac Diabetics

In general it is safer for these patients to live with a moderate hyperglycæmia and have glycosuria from time to time, than for them to be controlled more strictly. The myocardium may depend for its nutrition upon hyperglycæmia compensating for the deficient coronary flow.

Diets should restrict foods of high cholesterol content, *e.g.* eggs, cream, butter, cheese. When heart failure occurs, insulin may need to be increased as the basal metabolic rate rises, especially as fever may coexist. The ferric chloride test should be preferred to Rothera's test. If it is positive, treatment of the diabetes should take preference over the cardiac condition. Increased fluids, insulin and glucose (1 g. per unit of insulin) will then be required. Warmth and rest are essential from all points of view. The combination of coronary thrombosis with diabetic coma is very desperate, and treatment should be directed to the coma. The problem of fluid intake in congestive heart failure is most difficult. It is better to risk the onset of œdema in severe diabetes than to allow progressive hyperglycæmia and ketosis. Consequent œdema can be dispersed later with mercurial diuretics. If coma is present intravenous fluid must be given, signs of dehydration indicating a lack of 10–12 pints [about 5·7 to 7·2 litres] of fluid.

REFERENCE

Root, H. F., Bland, E. F., Gordon, W. H., & White, P. D. (1939), *J. Amer. med. Ass.*, **113**, 27

BLOOD PRESSURE AND OLD AGE

by T. H. Howell, *British Heart Journal*, **4**, 143–148, October 1942

Investigators in the past have agreed that a rise in blood pressure is customary after the age of 60, the peak being reached between 75 and 89, but falling thereafter. In the present paper from the *Royal* Hospital, Chelsea [a residential establishment for superannuated Army Pensioners], the author attempts to find some relationship between blood pressure, arteriosclerosis, mental state, and physical condition.

Conditions and Standards. The subjects were 120 tough veterans of the British professional Army aged 65 to 92. Each was classified by blood pressure, state of arteries, renal concentration power, mental state, and physical condition. Weekly estimations of the blood pressure were done, and variations were common. Infections, especially when accompanied by diarrhœa or pyrexia, caused a fall in pressure. Pressure also fell before death, particularly if due to cancer or cardiac failure.

Blood Pressure. A systolic pressure of over 160 mm. was taken as definitely high ; this occurred in 42% of the total, and included many of the fittest men in the establishment. Only 8 subjects had a systolic pressure of more than 200 mm. Of those with a high pressure, 33 were active physically and only 12 were slightly incapacitated out of a total of 50. Only 6 subjects had a systolic pressure regularly below 115 mm., and all but 1 of these died of cancer or cardiac failure.

Arteriosclerosis and Mental Condition. Each subject was classified according to the state of his arteries as slightly (+) moderately (++) or considerably (+++) thickened. In no case were the vessels soft. The mental condition was assessed as normal adult, normal senile, or impaired.

The author's findings are summarised in the following tables :—

Arteriosclerosis and Blood Pressure

Arterial Thickening	*Total Number*	*Raised Blood Pressure*	*Lowered Blood Pressure*	*Mentally Impaired*
Slight . .	13	16%	24%	8%
Moderate . .	80	38%	4%	22%
Marked . .	27	66%	20%	32%

Mental Condition and Circulation

Mental Standard	*Total Number*	*With raised Blood Pressure*	*Arterial Thickening* +	++	+++
Adult . .	15	73%	33%	67%	—
Senile . .	78	33%	10%	66%	24%
Impaired .	27	50%	4%	66%	30%

Age, Arteriosclerosis, and Raised Blood Pressure

Age	*Total Number*	*Arteries* +	++	+++	*High Blood Pressure*
Below 70 .	26	20%	65%	15%	40%
70–74 .	33	15%	70%	15%	37%
75–79 .	34	6%	76%	18%	50%
Over 80 .	27	4%	52%	44%	33%

Conclusions. A systolic blood pressure above 160 mm. is common in old age, occurring in half to a third of the cases. Amongst the oldest patients the fittest were those with a high pressure. This suggests that a high pressure is needed to offset the increasing arteriosclerosis of old age. Marked thickening of arteries with a low pressure is associated with a poor physical condition, whilst a raised pressure and thickened arteries are compatible with relative fitness and activity. No man with very thickened arteries had adult intelligence, and no man with impaired intellect had other than at least moderate arteriosclerosis. Measurement of the specific gravity of the urine revealed nothing of significance.

It appears that as arteriosclerosis increases, a greater blood pressure is needed to prevent ischæmia of vital organs. Thus senile hypertension may be compensatory in nature. In its absence, vital ischæmia results in impaired function, either mental or physical.

This Bulletin is published by the British Council. Requests for further information about any of the investigations reported should be addressed to The British Council, Medical Department, 3 Hanover Street, London, W. 1, England

Printed by Spottiswoode, Ballantyne and Co. Ltd., Colchester, London and Eton